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Editorial

# Disease Ecology in the Face of Climate Change

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Global climate change has the potential to dramatically affect human health, especially impacting dynamics of parasites and infectious diseases. Rising temperatures and changes in rainfall patterns will alter the biogeographic patterns of microbes, insect vectors, animal reservoirs and susceptible humans and change the prevalence of infectious disease [1].

However, the effects of climate change and disease have rarely been considered together due to the difficulty in disentangling the multiple variables associated with parasite and pathogen diversity, host population dynamics, and short-term versus long-term environmental variation [2-5].

Food-borne infections follow seasonal patterns that have been associated with climate variability (increased temperatures, heat waves, and flooding) [6-7]. In addition, water-borne infectious diseases have been shown to be affected by seasonality [8]. During times of drought, that have been shown to have a higher probability in climate change models [9], water scarcity results in poor sanitation, and much of the population can be exposed to potentially contaminated water [10]. Colwell and Huq [11] studied the incidence of cholera and found significant relationship between ocean levels and sea surface temperature, both are expected to rise under current climate forecasts.

Climate change may affect zoonoses either by changing the range of reservoirs or vectors, or by prolonging transmission cycles [1]. Specifically, rainfall and temperature affect mosquito vector abundance, biting rates and parasite development within vectors that can have profound influence on malaria [12], dengue [13], and West Nile virus [14].

Not only does climate change alter disease ecology, but it also influences pathogen and parasite evolution. Anthropogenic disturbanceshave been shown to negatively impact microbial communities [15]. The stress applied to microbial communities can be sufficient to promote horizontal gene transfer [16]. The strategies employed by microbes to resist environmental stressors can result in transfer of antibiotic resistance elements [17]. However, Horizontal Gene Transfer (HGT) is also responsible for the creation of novel pathogens and parasites through interspecific transfer of disease elements and toxins known as 'pathogenicity islands' [18-20]. The first cases of HGT in eukaryotes were associated with parasite evolution [21,22]. Whether through acquisition of antibiotic resistance, pathogenicity islands, or expanding to new metabolism [23] or host-specific environments [24], HGT is due in part to the fact that those genes that confer some evolutionary benefit are most likely to be found in other organisms already adapted to those circumstances [25].

Four key areas of research are needed in order to provide a better picture of the future of disease ecology; standardized metadata collection, improved baseline measurements, model building, and open access.

Metadata standards: what information should be collected with disease ecology?

Similar to the Ecological Metadata Language (EML) project based on work by the Ecological Society of America to manage

data associated, in this case, with disease ecology. (see http://knb. ecoinformatics.org/index.jsp). Since this is a community oriented endeavor, more acceptance and encouragement of standards by publishers would benefit a building of current disease patterns for baseline measurements.

Baseline measurements: what are current disease prevalence patterns, and what resolution of strain identification is needed?

What are the biotic and abiotic factors that influence disease and what are the biogeographic patterns of different strains? In a recent study, Hendriksen et al. [26] identified the source of the cholera outbreak in Haiti as a group of Nepalese peacekeeping troops. A rebuttal, however, was published, which pointed out that South Asia, instead of Nepal, may be the origin of the Haitian cholera outbreak strains, stating that proper attribution of an outbreak to a source strain cannot be accomplished by an exclusionary approach [27]. How do we improve disease detection and biogeographic patterns?

# **Model Building**

Models that can couple the metadata and baseline measurements to detect patterns in climate and disease are essential for understanding disease ecology and predicting future outbreaks associated with changing climate.

### **Open Access**

Restriction of information on disease ecology and the implications of climate change is morally unpalatable at best. Over the past few years, the number of high quality open access targets for publication has exploded. Publication in journals such as the Journal of Bacteriology and Parasitology provides an excellent platform for the studies needed to resolve complexity of disease ecology in the face of climate change.

#### References

- Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, et al. (2002) Climate warming and disease risks for terrestrial and marine biota. Science 296: 2158-2162.
- Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, et al. (2008) Global trends in emerging infectious diseases. Nature 451: 990-993.
- Lafferty KD, Porter JW, Ford SE (2004) Are diseases increasing in the ocean? Annu Rev Ecol Evol Syst 35: 31-54.
- Lafferty KD (2009) The ecology of climate change and infectious diseases. Ecology 90: 888-900.

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- Rohr JR, Raffel TR, Romansic JM, McCallum H, Hudson PJ (2008) Evaluating the links between climate, disease spread, and amphibian declines. Proc Natl Acad Sci USA 105: 17436-17441.
- Bentham G, Langford IH (2001) Environmental temperatures and the incidence of food poisoning in England and Wales. Intl J Biometeorol 45: 22-26.
- Kovats RS, Campbell-Lendrum D, Matthies F (2005) Climate change and human health: estimating avoidable deaths and disease. Risk Anal 25: 1409-1418.
- Schijven J, Rijs GB, de Roda Husman AM (2005) Quantitative risk assessment of FMD virus transmission via water. Risk Anal 25: 13-21.
- 9. Hansen J, Sato M, Ruedy R (2012) Perception of climate change. Proc Natl Acad Sci U S A.
- Semenza JC, Suk JE, Estevez V, Ebi KL, Lindgren E (2011) Mapping climate change vulnerabilities to infectious diseases in Europe. Environ Health Perspectg 120: 385-392.
- Colwell RR, Huq A (1998) Global microbial ecology: biogeography and diversity of Vibrios as a model. J Appl Microbiol 85: 134S-137S.
- 12. Hoshen MB, Morse AP (2004) A weather-driven model of malaria transmission. Malar J 3: 32.
- Watts DM, Burke DS, Harrison BA, Whitmire RE, Nisalak A (1987) Effect of temperature on the vector efficiency of Aedes aegypti for dengue 2 virus. Am J Trop Med Hyg 36: 143-152.
- Campbell GL, Marfin AA, Lanciotti RS, Gubler DJ (2002) West Nile virus. Lancet Infect Dis 2: 519-529.
- Parnell JJ, Crowl TA, Weimer BC, Pfrender ME (2009) Biodiversity in microbial communities: system scale patterns and mechanisms. Mol Ecol 18: 1455-1462.
- Parnell JJ, Rompato G, Latta LC 4<sup>th</sup>, Pfrender ME, Van Nostrand JD, et al. (2010) Functional biogeography as evidence of gene transfer in hypersaline microbial communities. PLoS One 5: e12919.

- 17. Praveenya P, Brijesh KS, Sathish KD, Purvi K (2012) Antibiotic resistance creating new epoch. J Bacteriol Parasitol 3: 1.
- Hacker J, Blum-oehler G, Muhldorfer I, Tschape H (1997) Pathogenicity islands of virulent bacteria: structure, function, and impact on microbial evolution. Mol Microbiol 23: 1089-1097.
- Friesen TL, Stukenbrock EH, Liu Z, Meinhardt S, Ling H, et al. (2006) Emergence of a new disease as a result of interspecific virulence gene transfer. Nat Genet 38: 953-956.
- 20. Oliver RP, Solomon PS (2008) Recent fungal diseases of crop plants: is lateral gene transfer a common theme? Mol Plant Microbe Interact 21: 287-293.
- 21. Andersson JO (2005) Lateral gene transfer in eukaryotes. Cell Mol Life Sci 62: 1182-1197.
- Richards TA, Hirt RP, Williams BA, Embley TM (2003) Horizontal gene transfer and the evolution of parasitic protozoa. Protist 154: 17-32.
- de Koning AP, Brinkman FS, Jones SJ, Keeling PJ (2000) Lateral gene transfer and metabolic adaptation in the human parasite Trichomonas vaginalis. Mol Biol Evol 17: 1769-1773.
- Fast NM, Law JS, Williams BA, Keeling PJ (2003) Bacterial catalase in the microsporidian Nosema locustae: implications for microsporidian metabolism and genome evolution. Eukaryot Cell 2: 1069-1075.
- Keeling PJ (2009) Functional and ecological impacts of horizontal gene transfer in eukaryotes. Curr Opin Genet Dev 19: 613-619.
- Hendriksen RS, Price LB, Schupp JM, Gillece JD, Kaas RS, et al. (2011) Population genetics of Vibrio cholerae from Nepal in 2010: evidence on the origin of the Haitian outbreak. MBio 2: e00157-11.
- Keim PS, Aarestrup FM, Shakya G, Price LB, Hendriksen RS, et al. (2011) Reply to "South Asia instead of Nepal may be the origin of the Haitian cholera outbreak strain". MBio 2: e00245-11.

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